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=> d his

     (FILE 'HOME' ENTERED AT 14:36:57 ON 03 SEP 2003)

     FILE 'REGISTRY' ENTERED AT 14:37:04 ON 03 SEP 2003
L1             1 S 287714-41-4/RN

     FILE 'HCAPLUS' ENTERED AT 14:37:28 ON 03 SEP 2003
L2           101 S L1
L3             0 S L2 AND PRD<199902
L4             0 S L2 AND PD<19990201
L5             1 S L2 AND PRD<19990201

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     14:42:56 ON 03 SEP 2003
L6            74 S L2

     FILE 'REGISTRY' ENTERED AT 14:44:03 ON 03 SEP 2003
                E FENOFIBRATE/CN
L7             1 S E3

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     14:44:32 ON 03 SEP 2003
L8             2 S L6 AND L7
L9             0 S L6 AND 19990201
L10            1 S L6 AND 1999?
L11            0 S L6 AND 1998?
L12            0 S L6 AND 1997?
L13            3 S L8 OR L10
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This one may be helpful

There's no convenient way to limit by date in the "other databases". These were "false drops"

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=> d que stat l5
L1             1 SEA FILE=REGISTRY ABB=ON  287714-41-4/RN
L2           101 SEA FILE=HCAPLUS ABB=ON  L1
L5             1 SEA FILE=HCAPLUS ABB=ON  L2 AND PRD<19990201

=> d ibib abs hitrn l5 1-1

L5   ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2003:633275  HCAPLUS
TITLE:                    Novel anticholesterol compositions and method for
                          using same
INVENTOR(S):              Dudley, Robert; Liao, Shutsung; Song, Ching
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 13 pp., Cont.-in-part of U.S.
                          Ser. No. 137,695.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   8
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     US 2003153541      A1   20030814       US 2002-174934   20020619 <--
     WO 9922728         A1   19990514       WO 1998-US23041  19981030 <--
         W:  AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
             DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE,
             KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW,
             MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR,
             TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
             FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
             CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     US 6576660         B1   20030610       US 2000-530443   20000428 <--
     US 2002107233      A1   20020808       US 2002-72128    20020208
     US 2002193357      A1   20021219       US 2002-137695   20020502
PRIORITY APPLN. INFO.:                  US 1997-63770P   P  19971031 <--
                                        WO 1998-US23041  W  19981030 <--
                                        US 1999-131728P  P  19990430
                                        US 2000-530443   A2 20000428
                                        US 2000-560236   A2 20000428
                                        US 2001-267493P  P  20010208
                                        US 2001-288643P  P  20010503
                                        US 2001-348020P  P  20011108
                                        US 2002-72128    A2 20020208
                                        US 2002-137695   A2 20020502
AB   Disclosed are compns., methods, combinations, and kits for treating a
     disorder related to elevated serum cholesterol concn., for example,
     atherosclerosis, elevated LDL plasma levels, low HDL plasma levels,
     hypertriglyceridemia, hyperlipidemia, hypertension, hypercholesterolemia,
     cholesterol gallstones, lipid storage diseases, obesity, and diabetes.
     The compns., methods, combinations, and kits of the present invention are
     pharmaceutical compns. comprising at least two of an LXR receptor
     modulator, a therapeutically effective amt. of a catechin, and/or a
     therapeutically effective amt. of a lipid regulating agent, such as a
     HMG-CoA reductase inhibitor, a fibric acid deriv., niacin, a bile-acid
     sequestrant, an absorption inhibitor, probucol, raloxifene and its
     derivs., an azetidinone compd., and an unsatd. omega-3 fatty acid.
IT   INDEXING IN PROGRESS
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IT **287714-41-4**, Rosuvastatin
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(anticholesterol compns. contg. LXR modulators and lipid regulating agents)

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=> d que stat l13
L1             1 SEA FILE=REGISTRY ABB=ON  287714-41-4/RN
L2           101 SEA FILE=HCAPLUS ABB=ON  L1
L6            74 SEA L2
L7             1 SEA FILE=REGISTRY ABB=ON  FENOFIBRATE/CN
L8             2 SEA L6 AND L7
L10            1 SEA L6 AND 1999?
L13            3 SEA L8 OR L10
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L13 ANSWER 1 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:374169 BIOSIS
DOCUMENT NUMBER: PREV200300374169
TITLE: Comparison of the dose-response relationships of 2 lipid-lowering agents: A Bayesian meta-analysis.
AUTHOR(S): Berry, Donald A. (1); Berry, Scott M.; McKellar, John; Pearson, Thomas A.
CORPORATE SOURCE: (1) Department of Biostatistics, University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Blvd, Unit 447, Houston, TX, 77030-4009, USA: dberry@mdanderson.org USA
SOURCE: American Heart Journal, (June 2003, 2003) Vol. 145, No. 6, pp. 1036-1045. print.
ISSN: 0002-8703.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Background: Comparing the dose-response of a new drug to that of a previously studied drug can aid in understanding their relative potencies. Two dose-finding studies addressed the effect of a new drug, rosuvastatin, on its ability to decrease low-density lipoprotein cholesterol (LDL-C) levels. One of these studies included 2 doses of atorvastatin, and substantial additional information is available in the literature about the effect of atorvastatin on LDL-C level lowering. Methods: The 2 dose-finding studies of rosuvastatin considered otherwise healthy patients who had hypercholesterolemia. Comparable studies of atorvastatin were identified via a MEDLINE search in December **1999**. Multiple reviewer consensus identified 15 of 41 studies on atorvastatin published since 1996 that met these selection criteria: reporting of LDL-C level change from baseline at least 6 weeks after treatment initiation, doses administered, and treatment group sizes. Eligible populations had clinical evidence of hypercholesterolemia. We excluded studies with patients who had severe illness or a previous history of transplantation. Data extraction of the mean, sample sizes, and SDs (or CIs) by dose was carried out independently by multiple reviewers. We combined the results from the various studies with Bayesian hierarchical modeling and analyzed them with Markov chain Monte Carlo techniques. Results: Combining this study and literature results substantially increased the power to compare the dose-response relationships of rosuvastatin and atorvastatin. Rosuvastatin reduced LDL-C level by an estimated 10 to 17 percentage points more than atorvastatin when both were given at the same dose. Approximately one quarter of the dose of rosuvastatin achieved about the same magnitude of LDL-C level reduction as atorvastatin at dosages as high as 80 mg. This finding does not imply a 4-fold difference in efficacy overall and specifically does not describe the results at higher dosage levels. Conclusions: Bayesian meta-analysis of results from related studies allows the comparison of the dose-response relationships of 2 drugs, better estimates of a particular dose-response relationship within an individual study, and the expression of relative benefits (of dose and drug) in terms of probabilities. Explicitly comparing a study's results with historical data using Bayesian meta-analysis allows clinicians to view the study in

the larger context of medical research.

L13 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:230319 BIOSIS
DOCUMENT NUMBER: PREV200300230319
TITLE: An open-label, randomized, three-way crossover trial of the effects of coadministration of rosuvastatin and fenofibrate on the pharmacokinetic properties of rosuvastatin and fenofibric acid in healthy male volunteers.
AUTHOR(S): Martin, Paul D. (1); Dane, Aaron L.; Schneck, Dennis W.; Warwick, Michael J.
CORPORATE SOURCE: (1) AstraZeneca, Mereside, Alderley Park, Macclesfield, Cheshire, SK10 4TG, UK: paul.martin@astrazeneca.com UK
SOURCE: Clinical Therapeutics, (February 2003, 2003) Vol. 25, No. 2, pp. 459-471. print.
ISSN: 0149-2918.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Background: Rosuvastatin and fenofibrate are lipid-regulating agents with different modes of action. Patients with dyslipidemia who have not achieved treatment targets with monotherapy may benefit from the combination of these agents. Objective: The effect of coadministration of rosuvastatin and fenofibrate on the steady-state pharmacokinetics of rosuvastatin and fenofibric acid (the active metabolite of fenofibrate) was assessed in healthy volunteers. Methods: This was an open-label, randomized, 3-way crossover trial consisting of three 7-day treatment periods. Healthy male volunteers received one of the following treatment regimens in each period: rosuvastatin 10 mg orally once daily; fenofibrate 67 mg orally TID; and rosuvastatin+fenofibrate dosed as above. The steady-state pharmacokinetics of rosuvastatin and fenofibric acid, both as substrate and as interacting drug, were investigated on day 7 of dosing. Treatment effects were assessed by construction of 90% CIs around the ratios of the geometric least-square means for rosuvastatin+fenofibrate/rosuvastatin and rosuvastatin+fenofibrate/fenofibrate for the area under the plasma concentration-time curve (AUC) and maximum plasma concentration (derived from analysis of variance of log-transformed parameters). Results: Fourteen healthy male volunteers participated in the study. When rosuvastatin was coadministered with fenofibrate, there were minor increases in the AUC from 0 to 24 hours and maximum concentration (Cmax) of rosuvastatin: the respective geometric least-square means increased by 7% (90% CI, 1.00-1.15) and 21% (90% CI, 1.14-1.28). The pharmacokinetic parameters of fenofibric acid were similar when fenofibrate was dosed alone and with rosuvastatin: the geometric least-square means for fenofibric acid AUC from 0 to 8 hours and Cmax decreased by 4% (90% CI, 0.90-1.02) and 9% (90% CI, 0.84-1.00), respectively. The treatments were well tolerated alone and in combination. Conclusion: Coadministration of rosuvastatin and fenofibrate produced minimal changes in rosuvastatin and fenofibric acid exposure.

L13 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:568661 BIOSIS
DOCUMENT NUMBER: PREV200200568661
TITLE: Rosuvastatin alone and in combination with fenofibrate in hyperlipidaemic patients with type 2 diabetes.
AUTHOR(S): Durrington, P. (1); Hamann, A.; Tuomilehto, J.; Smith, K.; Kallend, D.
CORPORATE SOURCE: (1) University of Manchester, Manchester UK
SOURCE: Diabetologia, (August, 2001) Vol. 44, No. Supplement 1, pp. A165. print.
Meeting Info.: 37th Annual Meeting of the European

Association for the Study of Diabetes Glasgow, Scotland, UK September 09-13, 2001 European Association for the Study of Diabetes
. ISSN: 0012-186X.
DOCUMENT TYPE: Conference
LANGUAGE: English